Volume 6 Number 1
CASE

Acute dystonia induced by quetiapine: a case report

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Abstract

Quetiapine is an atypical antipsychotic which has been shown to have lower incidence of extrapyramidal symptoms (EPS). In recent times, few case reports and case series have reported occurrence of akathisia, dystonia, and Parkinsonism with quetiapine; however, data are still sparse. To add to this literature, we report a case of 23 years old male who developed acute dystonia on quetiapine.

Gedam SR, Ghosh SS. Acute dystonia induced by quetiapine: a case report. Open Journal of Psychiatry & Allied Sciences.2015;6:59-61
Keywords: Movement Disorders. Antipsychotic Agents. Dopamine.
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Received on 20 June 2014. Revised on 15 August 2014. Accepted on 15 August 2014.

Introduction

Dystonia refers to a movement disorder characterised by simultaneous involuntary contraction of agonist and antagonist axial and appendicular muscles.[1,2] Acute dystonia frequently develops secondarily as a reaction to some drugs and generally in the early phases of treatment, and sometimes even after a single dose of particular antipsychotic drug.[3] Acute dystonia occurs in up to ten per cent of patients, especially young men receiving an antipsychotic. The symptoms include abnormal positioning of the head and neck (e.g., torticollis); spasm of jaw muscles (e.g., trismus); impaired swallowing, speaking, or breathing; thickened or slurred speech; tongue protrusion or dysfunction; deviated eyes in any direction (e.g., oculogyric crisis [OGC]); and abnormal positioning of limbs or trunk (e.g., opisthotonos).[4]

Among risk factors are taking antipsychotic drugs for the first time, as well as taking highly potent and high doses, history of head trauma, anxiety, being an elderly woman and a young man.[3] Patients may develop the syndrome within minutes of receiving injectable high-potency first generation antipsychotics (FGAs), but symptoms usually develop within days of starting or increasing the oral antipsychotic dose or reducing a medication used to treat extrapyramidal symptoms (EPS).[4] There are a few dystonia cases reported connected to quetiapine.[5-10] Quetiapine, an atypical antipsychotic, has low-to-moderate affinity for D1 , D2 , 5-HT1A , and 5-HT2A receptors, and moderate-to-high affinity for the a1 - and a2 -adrenergic receptors. Like other atypical antipsychotics, quetiapine has greater relative affinity for 5-HT2A receptors than for D2 receptors. Due to this receptor profile, quetiapine has been claimed to cause less EPS.[11,12] Here we report a case of 23 years old male who developed dystonia while on quetiapine.

Case report

A 23 years old male was brought to psychiatry clinic with the complaints of protrusion of tongue, spasm of jaw, hypersalivation, restlessness, insomnia, and difficulty in speech for last three days. As per the informant and available documentation, he was diagnosed as brief psychotic episode before two years for which he was admitted in psychiatry unit for around one month with complete remission. He was under regular treatment on risperidone 6 mg/day, trihexyphenidyl 2 mg/day, and diazepam 7.5 mg/day. After few months he developed tremors and rigidity on same medications. So he was switched to quetiapine (increased from 50 mg to 200 mg over a period of one month), trihexyphenidyl (increased from 2 mg to 6 mg over a period of one month), and clonazepam 3 mg/day, and reported improvement of EPS. Despite good compliance with medi-

cations he developed symptoms in the form of decreased sleep, smiling to self, social withdrawal, disorganised behaviour, decreased appetite, and severe socio-occupational dysfunction; so the dose of quetiapine was raised to 300 mg/day and diazepam 10 mg/day which he was receiving for last two and half months from current presentation. As there was no EPS the dose of trihexyphenidyl was decreased to 2 mg/day (decreased from 6 mg to 2 mg) during same period.

There was no past history suggestive of similar symptoms, medical disorder, or substance use. The paternal uncle had history of psychiatric illness responding significantly to treatment. The patient was thoroughly assessed, diagnosed as drug induced acute dystonia, and hospitalised in psychiatry unit for management. Injection promethazine hydrochloride 25 mg intramuscular (IM) was given and within 30 minutes he showed complete improvement in EPS. Laboratory tests including liver function tests, whole blood count, and routine biochemical tests revealed no abnormalities. The dose of quetiapine tapered down (reducing 50 mg/day) over a period of one week with no return of dystonia symptoms. In view of relapse of psychotic symptoms he was started on olanzapine which was gradually optimised according to symptoms.

Discussion

The mechanism of acute dystonia in humans is still unclear, but the connection between the serotonergic and dopaminergic systems seems to play a major role. In central dopaminergic systems two main reactions develop against dopaminergic blockage. The acute and short lasting reaction is the increase in dopamine's turnover. This is in keeping with the development of clinical period of acute dystonia. During this period blood drug level is not stable; it increases and more importantly it decreases rapidly. Secondly, slower and long acting one is the "post synaptic receptor super sensitivity".[13] These, two factors constitute basic principles of "mismatch" hypothesis. According to this hypothesis due to increased strial dopamine release induced by "increased turnover" blockade postsynaptic receptors in inconsistent and unbalanced way. As antipsychotic levels fall down this blockades disappear, and causes development of acute dystonia.[14,15]

The symptoms of medication-induced acute dystonia usually occur within hours to several days of starting, increasing or decreasing the dose of medication; severity usually decreases with rest and relaxation.[16] The appearance period of quetiapine induced dystonia and its relation with dose show differences in related reports.[5-10] Ghosh et al.[17]reported from Assam, India the first case of quetiapine-associated tardive OGC. Chaubey et al.[18] discussed tardive dyskinesia (TD) from the point of view of dental implications. Generally when there appears to be no organic defect in the base with quetiapine induced dystonia cases the dose is approximately 400-600mg/day with a duration of two to four weeks, but in the presence of a defect in the base (such as Parkinson's disease, head trauma) a very low dose such as 12.5 mg and a very short time, or rather high dose and a very short period of time dystonia is induced.[5-7]

In this patient manifestation of dystonia started while the dose of quetiapine increased to 300 mg/day and the dose of trihexyphenidyl decreased to 2 mg/day. His symptoms subsided after providing promethazine hydrochloride IM and quetiapine tapered down over a period of one week. These findings suggest that dystonia was a true side effect of quetiapine. As is evident from the available case reports, the major risk factors for the development of dystonia on quetiapine are history of EPS with other antipsychotic and a young aged man. These findings are in accordance with presenting case report. In such subjects, caution should be exercised while prescribing quetiapine and dose should be increased gradually and patients should be monitored closely.

Source of support: Nil. **Declaration of interest:** None.

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